Baicalein inhibits heparin-induced Tau aggregation by initializing Tau oligomer formation

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Video Byte

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Abstract

Alzheimer's disease (AD) is the leading cause of dementia in the aged population. The key feature of AD is the deposition of two different kinds of protein aggregates in the brain, and at the point of aggregate formation, treatment becomes difficult. A recent study focused on how to prevent the aggregation of one protein: the microtubule-associated protein Tau, which forms neurofibrillary tangles. Following up on research indicating that polyphenolic compounds can serve as neuroprotective agents, researchers analyzed the ability of the polyphenol Baicalein to inhibit the aggregation of Tau. In vitro, Baicalein blocked Tau aggregation and paired helical filament dissolution via an oligomer capture and dissociation mechanism. It also dissolved preformed mature fibrils of Tau, creating Tau oligomers, with no effect on the viability of neuronal cells. Although further studies are needed to confirm these effects in vivo, the results highlight the potential for Baicalein to serve as a targeted inhibitor of Tau pathology in AD.